Triethylene Glycol
(CAS# 112-27-6)

(Synonyms: 2,2'-(1,2-Ethanediylbis(oxy))bisethanol; 1,2-Bis(2-hydroxyethoxy)ethane; 2,2'-Ethylenedioxybis(ethanol); 2,2'-Ethylenedioxydiethanol; 2,2'-Ethylenedioxyethanol; 3,6-Dioxaoctane-1,8-diol; Bis(2-hydroxyethoxyethane); Di-beta-hydroxyethoxyethane; Ethanol, 2,2'-(1,2-ethanediylbis(oxy))bis-; Ethanol, 2,2'-(ethylenedioxy)di-; Ethylene glycol dihydroxydiethyl ether; Ethylene glycol-bis-(2-hydroxyethyl ether); Glycol bis(hydroxyethyl) ether; Trigen; Triglycol; TEG)

Triethylene Glycol Acute REL

Reference Exposure Level	**30.9 mg/m^3 (2 ppm) [Inhalation]**
Critical effects	Systemic
Hazard Index target	Nervous system

Triethylene Glycol 8-hour REL

Reference Exposure Level	**0.14 mg/m^3 (0.023 ppm) [Inhalation]**
Critical effects	Systemic
Hazard Index target	Nervous system

1 Physical and Chemical Properties

Physical form	clear, colorless liquid
Structural formula	H-(O-CH_2-CH_2)$_3$-OH
Molecular weight	150.17 g/mole
Density	1.126 g/cm^3 @ 20 ºC
Boiling point	285 ºC
Melting point	-7 ºC
Vapor pressure	0.00132 mm Hg @ 25 ºC
Flash point	177 ºC (closed cup)
Log K_{OW}	-1.98
Water solubility	fully miscible
Atmospheric half-life	3.8 hrs
Conversion factor	1 ppm = 6.14 mg/m^3

2 Production, Use, and Exposure

Triethylene glycol (TEG), prepared from ethylene oxide and ethylene glycol, is manufactured by forming an ether-ester of hydroxyacetic acid with glycol, followed by hydrogenation. TEG is used in cosmetics as a fragrance ingredient and as a viscosity-decreasing agent. TEG is also used in various plastics to increase pliability, in air sanitizers, in brake fluid, as a solvent and plasticizer in vinyl, polyester, and polyurethane resins, in dehydration of natural gas, as a humectant in printing inks, as an extraction solvent, and as a fungicide and solvent for nitrocellulose (CIR Expert Panel 2006). Although not supported by some manufacturers, TEG formulations have been used for the generation of smokes, mists or fogs for theatrical purposes (Ballantyne and Snellings 2007).

With normal manufacturing practices, emissions to wastewater and air are minimal although small amounts may be released from spills and cleaning operations. TEG can also enter the atmospheric, aqueous or terrestrial environment from its various end uses. The primary occupational exposures to TEG occur via skin contact during manufacturing. Occupational vapor exposure to TEG is expected to be low due to its low vapor pressure. TEG is not a skin irritant, but acute eye contact with the liquid may result in mild transient irritation. Consumer exposure occurs mostly from the use of cosmetics, cleaning products, brake fluid, and air sanitizers containing TEG. According to the U.S. Department of Health and Human Services Household Products Database, TEG is in brake fluid (liquid) at concentrations up to 25%, in oven cleaner (aerosol), and in various brands of air sanitizer or room deodorizer (aerosol) at concentrations up to 6%. Uses of these products can result in direct dermal and inhalation exposures, as well as indirect exposure through the ingestion of contaminated drinking water.

3 Pharmacokinetics and Metabolism

McKennis et al. (1962, cited by CIR Expert Panel 2006) administered single oral doses of 22.5 mg ^{14}C-TEG to four male albino rats weighing 112 to 145 g. Urine, feces, and expired air were collected in a metabolic chamber over a period of 5 days. The radioactivity recovered, measured as percent of the administered dose, was 0.8% to 1.2% in expired air, 2.0% to 5.3% in feces, and 86.1% to 94.0% in urine. The total recovery of the administered dose was 90.6% to 98.3%. In the same study, two female New Zealand white rabbits were given 200 or 2000 mg/kg TEG by stomach tube. Urine collected over the following 24 hours from the dosed animals contained 34.3% or 28% of the respective dose amount as unchanged TEG. One animal excreted 35.2% of the administered dose as a hydroxyl acid from TEG in the urine.

4 Acute Toxicity

Acute mammalian toxicity is summarized as follows:

Species/Route	LD_{50} or LC_{50}	Reference
Intraperitoneal – mouse	8.15 g/kg	Karel et al. (1947) cited in CIR Expert Panel (2006)
Intravenous – dog	> 4500 mg/kg	NIOSH (2003)
Intravenous – mouse	6500 mg/kg	NIOSH (2003)
Intravenous – rat	11700 mg/kg	NIOSH (2003)
Intravenous – rabbit	1900 mg/kg	NIOSH (2003)
Intravenous – rat Oral – rat	7.3 to 9.5 g/kg 15 to 22 g/kg	Budavari et al. (1989) cited in CIR Expert Panel (2006)
Oral – rat Oral – guinea pig Oral – mouse	22 g/kg 14.7 g/kg 18.5 g/kg	Smyth et al. (1941) cited in CIR Expert Panel (2006)
Oral – guinea pig	7900 mg/kg	NIOSH (2003)
Oral – mouse	> 18500 mg/kg	NIOSH (2003)
Oral – rabbit	8400 mg/kg	NIOSH (2003)
Oral – rabbit	9500 mg/kg	NIOSH (2003)
Inhalation (aerosol) – rat	> 4400 mg/m^3	Cascieri et al. (1991)
Inhalation (aerosol) – rat	> 3.9 mg/L	Union Carbide (1990) cited in CIR Expert Panel (2006)

Table adapted from Ballantyne and Snellings (2007) with additional information from CIR Expert Panel (2006).

Although there are no human studies on the oral toxicity of TEG, there are isolated cases of poisoning by swallowing TEG-containing products such as brake fluid. Vassiliadis et al. (1999) reported the case of a 23-year-old woman who intentionally ingested one gulp (volume not specified) of Caltex® brake fluid. According to the product's material safety data sheet (MSDS), the brake fluid contained 30.00-60.00% polyglycol ethers, 30.00-60.00% borate of triethylene glycol monomethyl ether, 30.00-60.00% polyglycol, 0-10.00% corrosion inhibitor, and 0-10.00% dye (CIR Expert Panel 2006). The patient presented with coma and metabolic acidosis and was treated with intravenous ethanol, resulting in a full recovery. The authors suggested that polymer homologs of ethylene glycol, such as TEG, are oxidized by alcohol dehydrogenase (ADH) to diacid and hydroxy acids, therefore treatment with a competing substrate of ADH such as ethanol was effective. In support of this, the authors cited a report by Borron et al. (1997), describing a 15-year-old female who ingested 200 ml of brake fluid containing 55% TEG and 10% diethylene glycol, who also presented with acidosis and was successfully treated with 4-methylpyrazole, a potent inhibitor of ADH (Vassiliadis et al. 1999).

5 Derivation of Acute REL (1-hour exposure)

This acute REL is derived from a study by Union Carbide (1990a), in which 4 groups of 5 male and 5 female Sprague-Dawley albino rats were exposed (whole body) once to an aerosol atmosphere of 2600, 3900, 5000, or 6700 mg/m^3 TEG for 4 hours. All 5 female rats in the 5000 mg/m^3 died within 2-3 days post-exposure, but when this exposure was repeated, no deaths were observed. No other deaths were observed at all dose levels. Clinical signs of toxicity observed for the 6700 and 5000 mg/m^3 groups on the day of exposure included a bright red discoloration of the eyes, ears, and feet, blepharospasm (spasmodic winking), and an absence of toe and tail pinch reflexes. In the repeat exposure of female rats to the 5000 mg/m^3 dose, audible respiration and decreased motor activity were observed in the first few days of the post-exposure period. A brown discoloration of the kidneys (2 males at the 5000 mg/m^3 dose) and dark red discoloration of the liver (1 female in the 3900 mg/m^3 group) were the only treatment-related macroscopic lesions observed in rats that were sacrificed at the end of the 2-week recovery period. No treatment-related microscopic lesions of the lungs or kidneys were found at the two highest doses. Based on the clinical signs of toxicity, the LOAEL is determined to be 5000 mg/m^3 and the NOAEL is 3900 mg/m^3. A value of $n = 3$ is used in extrapolating from an experimental exposure duration of greater than one hour to one hour. The interspecies uncertainty factor is adjusted to $2 * \sqrt{10}$ because the U.S. EPA Human Equivalent Concentration procedure is used as a partial adjustment for interspecies toxicokinetic differences. A regional gas dose ratio (RGDR) of 1 is used for gases with systemic effects, following U.S. EPA's recommendation that an RGDR of 1 be used when the relevant blood:air coefficients are unknown. For the intraspecies toxicokinetic uncertainty sub-factor, a value of 10 is used in consideration of the protection of children's health and sensitive subgroups. Default values of $\sqrt{10}$ are used for the interspecies and intraspecies toxicodynamic uncertainty sub-factors in the absence of data to indicate otherwise. As indicated below in the derivation of the 8-hr REL, the time-extrapolated concentration associated with the critical effects in this study gave a lower point of departure, and consequently a lower REL, than would be derived from the developmental study by Ballantyne and Snellings (2005). For this reason no additional uncertainty factors were applied.

Study	Union Carbide (1990a)
Study population	Sprague-Dawley albino rats
Exposure method	Whole-body inhalation
Exposure continuity	Once
Exposure duration	4 hours
Critical effects	Bright red discoloration of the eyes, ears, and feet; blepharospasm; absence of toe and tail pinch reflexes
LOAEL	5000 mg/m^3
NOAEL	3900 mg/m^3
Time-adjusted exposure	C^n * T = K, *n* = 3 (ten Berge et al. 1986)
Extrapolated concentration	6185 mg/m^3 $(3900^3 * 4)^{1/3}$
Human concentration adjustment	6185 mg/m^3 (RGDR = 1; systemic)
LOAEL uncertainty factor (UF_L)	1 (NOAEL observed)
Subchronic uncertainty factor	1 (not applicable for an acute REL)
Interspecies uncertainty factor	
Toxicokinetic (UF_{A-k})	2
Toxicodynamic (UF_{A-d})	√10
Intraspecies uncertainty factor	
Toxicokinetic (UF_{H-k})	10
Toxicodynamic (UF_{H-d})	√10
Cumulative uncertainty factor	**200**
Acute Reference Exposure Level	**30.9 mg/m^3**

RGDR: regional gas dose ratio

6 Derivation of 8-Hour RELs

6.1 Derivation of 8-Hour REL with Inhalation Study

We first derived an 8-hour REL from a study by Ballantyne et al. (2006), in which 4 groups of 10 male and 10 female Sprague-Dawley rats were exposed to an aerosol atmosphere of 0, 494, 2011, or 4824 mg/m^3 TEG for 6 hrs/day, for a total of 9 exposures over a period of 11 days. All rats died at the 4824 mg/m^3 concentration while the remainder of the animals all survived the lower concentrations. At 494 mg/m^3, female rats had a statistically significant increase in alkaline phosphatase activity, which is a biochemical indication of possible liver dysfunction, but this was not accompanied by histological evidence of liver injury. There were also signs of minor eye irritation, increased water consumption, and increased inorganic phosphorus levels in females for this dose group. The authors of this study noted that the low vapor pressure of TEG would result in significant wetting of the fur and contribute to ingestion by preening. Therefore, they conducted an additional nose-only inhalation study, under the same conditions, and found no statistically significant effects in exposed animals compared to controls at the high dose of 1036 mg/m^3. The whole-body exposure study was used in the derivation of this REL because oral exposure to TEG is relevant to humans when TEG-containing products are used in aerosol form. Furthermore, the eye irritation observed in the rats would have likely occurred irrespective of the ingestion of TEG by preening. Since a NOAEL was not determined in this study, a LOAEL uncertainty factor of 10 is applied. A value of *n* = 1 is used in extrapolating from an experimental exposure duration of less than 8 hours to an 8-hour level. To account for the possibility of long-term repeated exposures, a subchronic uncertainty factor of 10 is applied since the experimental exposure was < 8% of the expected lifetime of the species tested. The interspecies uncertainty factor is adjusted to 6 (2 * √10) because the U.S. EPA Human Equivalent Concentration procedure is used as a partial adjustment for interspecies toxicokinetic differences. A regional gas dose ratio of 1 is used for gases with systemic effects, following U.S. EPA's recommendation that an RGDR of 1 be used when the relevant blood:air coefficients are unknown. For the intraspecies toxicokinetic uncertainty sub-factor, a value of 10 is used in consideration of the protection of children's health and sensitive subgroups. Default values of √10 are used for the interspecies and intraspecies toxicodynamic uncertainty sub-factors in the absence of data to indicate otherwise. However, the cumulative uncertainty of 20,000 indicates that this is not an appropriate study for REL derivation.

Study	Ballantyne et al. (2006)
Study population	Sprague-Dawley rats
Exposure method	Whole-body inhalation
Exposure continuity	6 hrs/day, 9 days
Exposure duration	11 days
Critical effects	Alterations in serum chemistry
LOAEL	494 mg/m^3
NOAEL	Not observed
Time-adjusted exposure	$C^n * T = K$, $n = 1$ (ten Berge et al., 1986)
Extrapolated concentration	265 mg/m^3 (494 * 6/8 * 5/7)
Human concentration adjustment	265 mg/m^3 (RGDR = 1 systemic)
LOAEL uncertainty factor (UF_L)	10
Subchronic uncertainty factor	10
Interspecies uncertainty factor	
Toxicokinetic (UF_{A-k})	2
Toxicodynamic (UF_{A-d})	√10
Intraspecies uncertainty factor	
Toxicokinetic (UF_{H-k})	10
Toxicodynamic (UF_{H-d})	√10
Cumulative uncertainty factor	20,000
8-hour Reference Exposure Level	**13 μg/m^3**

RGDR: regional gas dose ratio

Since the cumulative UF of 20,000 indicated too much uncertainty in Ballantyne's study as the basis for an 8-hr REL, we derived an 8-hr REL using the same inhalation study by Union Carbide (1990a) as in the acute REL. This will be compared with a developmental study (Ballantyne and Snellings, 2005) in which the endpoint is fetotoxicity and the route of exposure is by oral gavage.

Study	Union Carbide (1990a)
Study population	Sprague-Dawley albino rats
Exposure method	Whole-body inhalation
Exposure continuity	Once
Exposure duration	4 hours
Critical effects	Bright red discoloration of the eyes, ears, and feet; blepharospasm; absence of toe and tail pinch reflexes
LOAEL	5000 mg/m^3
NOAEL	3900 mg/m^3
Time-adjusted exposure	$C^n * T = K$, $n = 1$ (ten Berge et al. 1986)
Extrapolated concentration	279 mg/m^3 (3900 * 4/8 * 1/7)
Human concentration adjustment	279 mg/m^3 (RGDR = 1 systemic)
LOAEL uncertainty factor (UF_L)	1 (NOAEL observed)
Subchronic uncertainty factor	10
Interspecies uncertainty factor	
Toxicokinetic (UF_{A-k})	2
Toxicodynamic (UF_{A-d})	√10
Intraspecies uncertainty factor	
Toxicokinetic (UF_{H-k})	10
Toxicodynamic (UF_{H-d})	√10
Cumulative uncertainty factor	2000
8-hour Reference Exposure Level	**0.14 mg/m^3 (0.023 ppm)**

As described in the acute REL, a NOAEL of 3900 mg/m^3 was observed. Adjustment of this value for an 8-hour exposure repeated daily gave 279 mg/m^3. The interspecies uncertainty factor is 2 for toxicokinetic variability because the U.S. EPA Human Equivalent Concentration procedure is used as a partial adjustment for interspecies differences. Interspecies toxicodynamic variation is addressed with a UF of √10. A regional gas dose ratio (RGDR) of 1 is used for gases with systemic effects, again following U.S. EPA's recommendation. For the intraspecies toxicokinetic uncertainty, a value of 10 is used for the protection of children's health and sensitive subgroups. Default values of √10 are used for the interspecies and intraspecies toxicodynamic uncertainty sub-factors in the absence of data to indicate otherwise. The cumulative UF is 2,000 and gives an 8-hr REL of 0.14 mg/m^3.

To ensure that the 8-hr REL derived above is protective of potentially more sensitive life stages, a REL was also derived based on a developmental study (Ballantyne and Snellings, 2005) (Section 6.2). Since the fetotoxicity endpoint is a function of exposure only during gestation and TEG is non-accumulating, the exposure is considered chronic for the fetus. Comparison with the 8-hr REL of 0.14 mg/m^3, a REL of 39.4 mg/m^3 indicates that the primary chronic response of fetotoxicity is more than 100 times less sensitive than the acute irritant effects from aerosol inhalation of TEG. As such, an 8-hour REL derived from the inhalation study used for the acute REL (Union Carbide 1990a) should be protective against the fetotoxic effects resulting from the repeat exposures to the fetus.

6.2 Derivation of 8-Hour REL with Route-to-route Extrapolation

Study	Ballantyne and Snellings (2005)
Study population	CR1:CD®-1 (ICR) BR albino mice
Exposure method	Oral gavage
Exposure continuity	Once daily
Exposure duration	Gestational days 6-15
Critical effects	Fetotoxicity – reduced ossification
LOAEL	5630 mg/kg-d
NOAEL	563 mg/kg-d
LOAEL uncertainty factor (UF_L)	1 (NOAEL observed)
Subchronic uncertainty factor	Not applicable
Interspecies uncertainty factor	
Toxicokinetic (UF_{A-k})	√10
Toxicodynamic (UF_{A-d})	√10
Intraspecies uncertainty factor	
Toxicokinetic (UF_{H-k})	√10 (fetus)
Toxicodynamic (UF_{H-d})	√10
Cumulative uncertainty factor	100
Oral dose	5.63 mg/kg-d (563 mg/kg-d/100)
Route-to-route extrapolation factor	70 kg/20 m^3/d
Chronic to 8-hour adjustment	(20 m^3/d)/(10 m^3/d)
8-hour Reference Exposure Level	**39.4 mg/m^3** (5.63 mg/kg-d*3.5 kg/m^3d*2)

In a developmental study using oral gavage, Ballantyne and Snellings (2005) administered 0, 563, 5630, or 11260 mg/kg-d TEG to timed-pregnant CD-1 mice during gestation days 6-15. The LOAEL of 5630 mg/kg-d was based on statistically significant reduced fetal body weights and an increased incidence of poorly ossified frontal and supraoccipital bones. The authors indicated a NOAEL of 563 mg/kg-d for fetotoxicity. Since the fetotoxicity endpoint is a function of exposure only during gestation and TEG is non-accumulating, the exposure is considered chronic for the fetus and an uncertainty factor to account for differences between subchronic and chronic exposures is not applied. Default values of √10 are used for interspecies and intraspecies toxicokinetic and toxicodynamic variability. In converting the oral dose to an air concentration, it is assumed that the efficiency of TEG absorption is the same between an oral dose and inhalation. The route-to-route conversion factor assumes that a 70 kg adult male breathes 20 m^3 air/d. The chronic to 8-hour adjustment applied here is based on the assumption that half of the 20 m^3 of air breathed in any 24-hour period is breathed while active at work. The resultant REL is therefore the oral dose multiplied by the route-to-route extrapolation factor and by the chronic to 8-hour adjustment.

7 Other Toxicity

Chronic Toxicity

Robertson et al. (1947) exposed both rats and Macaque rhesus monkeys to TEG orally and by inhalation of vapor. Thirty-six rats were exposed to a supersaturated vapor atmosphere of TEG continuously for 13 months and 3 groups of 8 rats were given TEG in their drinking water for the same amount of time, at concentrations calculated to be 35, 80, and 700 times the maximum quantity a rat could inhale in vapor form if kept in a saturated atmosphere for 24 hrs. The rats in both the oral and inhalation studies were allowed to breed and all the offspring appeared to be normal in every way, and gained weight just as rapidly as the controls of the same age. There were no apparent differences between the rats exposed to TEG vapor or TEG in drinking water compared to controls in postmortem findings. Seventeen monkeys were exposed to a continuous supersaturated fog of TEG for 13 months. The main differences between TEG-exposed and control animals were an overall slower and lesser degree of weight gain and a browning of the facial skin in the exposed animals. Otherwise, the monkeys were all very active, ate well, and had smooth glossy coats, with no other differences between treatment and control groups. Eight monkeys were also given TEG orally by adding it to egg nog which was always readily consumed. The daily oral doses were calculated as representing approximately 50 and 100 times the amount which a monkey could inhale in 24 hours in an atmosphere saturated with TEG. The weight gain in these monkeys was a little less than the control group and examinations of blood and urine yielded practically identical results with the controls except that the exposed monkeys showed less anemia at the end of the test period. To address the decrease in weight gain and the browning of the skin, the authors exposed another group of monkeys to a 65-75% saturated vapor atmosphere of TEG for 10 months. Under these conditions, none of the monkeys showed skin discoloration and after the first month, the TEG-exposed monkeys showed a slight but consistently greater weight gain than the controls. There were no pathological changes attributable to the exposure to TEG.

Epidemiology

Because of its bactericidal properties, TEG was tested in the past for use in controlling air-borne infections. Bigg et al. (1945) tested the effectiveness of TEG in reducing the incidence of air-borne infections by treating barracks, housing 320 men, with a concentration of 0.0025 to 0.003 mg TEG per liter of air for 6 week periods. Although toxicity was not the focus of the study, the authors noted that frequent interrogation of the men concerning possible effects of the vapor elicited no evidence of irritation of the respiratory tract. There were no other reports of symptoms arising from the vapor exposure.

Loosli et al. (1947) investigated the effectiveness of TEG in controlling infections in an infants' ward from November 1945 to April 1946. The concentration of TEG vapor in the air of the test ward varied from 55% to 70%. The consumption of TEG was approximately 200 ml per day. There were no major complaints from the attendants, nurses, or doctors who worked on the ward from a few days to several months. One or two nurses complained of headaches only when a visible TEG fog was present. Infants living on the test ward from a few days to several weeks displayed no evidence of frank toxic effects on the respiratory tract or skin.

In 1990, the National Institute for Occupational Safety and Health (NIOSH 1994) was asked to evaluate the possible health effects associated with the use of theatrical smoke in Broadway productions. Triethylene glycol was detected in only one production, at levels ranging from < 0.04 to 3.7 mg/m^3. NIOSH concluded that there was no evidence that theatrical smoke, at the levels found in the theaters studied, is a cause of occupational asthma in performers. However, they stated that some constituents of theatrical smoke, such as aerosolized glycols, could have irritative or mucous membrane drying properties in some individuals.

Reproductive and Developmental Toxicity
In addition to the developmental study used above for comparison with the derived 8-hour REL, other studies also suggest fetotoxicity with TEG exposure. The CIR Expert Panel (2006) reported a study by Union Carbide in which pregnant CD-1 mice were exposed to 0, 0.5, 5, or 10 ml/kg/day (n = 30 mice/group) undiluted TEG by oral gavage daily on gestation days 6 through 15. Maternal clinical signs observed in the 10 ml/kg/day group included hyperactivity with audible and rapid respiration and necropsy revealed that relative, but not absolute, kidney weights were increased in the high-dose group. The sum of fetal body weights per litter were significantly reduced in the 5 and 10 ml/kg/day groups, which coincided with decreased ossification of various bones at these doses.

In another Union Carbide study cited by the CIR Expert Panel (2006), Sprague-Dawley rats were exposed to 0, 1, 5, or 10 ml/kg/day (n = 55 rats per group) undiluted TEG by oral gavage on gestation days 6 through 15. The results of this study were similar to their study with CD-1 mice in that there was some maternal toxicity at the 10 ml/kg/day dose, evidenced by audible respiration, urine stains, periocular encrustation, perioral wetness, and at necropsy, an increase in relative kidney weight. Fetal body weights per litter were also decreased in the 10 ml/kg/day group, and there was an increase in the incidence of one skeletal variation (bilobed thoracic centrum no. 10) at this dose.

In addition to the mouse data used in the oral gavage study described above, Ballantyne and Snellings (2005) also dosed pregnant CD rats by gavage daily with undiluted TEG over gestational days 5-15 at concentrations of 0 (water control), 1126, 5630, or 11,260 mg/kg/day for rats compared with 0, 563, 5630, or 11,260 mg/kg/day for mice. Rat dams had reduced body weight and increased water consumption, and fetal body weights were reduced at 5630 mg/kg/day. Rat fetuses exhibited a pattern of delayed ossification in the thoracic region at 11,260 mg/kg/day. Mice had clinical signs and increased relative kidney weight at 11,260 mg/kg/day. Fetal body weights of mice were reduced at 5630 mg/kg/day. Mouse fetuses had delayed ossification in the frontal and supraoccipital bones, cervical region, hind limb proximal phalanges and reduced caudal segments at 11,260 mg/kg/day, and delayed ossification in the skull bones at 5630 mg/kg/day.

8 Environmental Fate

The estimated atmospheric photodegradation (reaction with hydroxyl radicals) half-life of TEG is 3.53 hours, based on 12 hours of sunlight/day and an average hydroxyl radical concentration of 5 x 10^5 OH/cm^3. Glycols have no hydrolysable groups and are generally not susceptible to hydrolysis in water under neutral conditions at ambient temperatures. An estimated soil-sediment coefficient (K_{OC}) of 10 indicates high soil mobility for TEG, which is not expected to undergo hydrolysis in moist terrestrial environments and/or direct photolysis on sunlit soil surfaces. The estimated Henry's Law Constant of 3.16 x 10^{-11} atm-m^3/mole for TEG indicates that it has limited potential to partition from water to air. Rapid biodegradation is likely to be the most important removal mechanism of TEG from the environment, with complete degradation of 10 mg/l complete within 7-11 days. With a predicted bioconcentration factor of 3.16 and Log K_{OW} of 1.75, TEG is not expected to bioaccumulate. TEG is therefore unlikely to persist in the environment (Ballantyne and Snellings 2007).

9 References

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BMDS Model Run Output File

NCTR Model Poor Ossification.pdf